MILD DEHYDRATION INDUCES MITRAL VALVE PROLAPSE IN FEMALE VOLUNTEERS WITH PRIOR NORMAL CARDIAC FINDINGS. Martha Eicher B.A., Daniela Lax M.D., Stanley J. Goldberg M.D., FACC. Univ. of Arizona, Tucson, Arizona.

The purpose of our study was to evaluate whether mitral valve prolapse (MVP) might vary with alterations in LV volume. We tested the hypotheses that 1) MVP can be induced in normal women, of characteristic body habitus, following diuresis and 2) any MVP changes thus produced can be reversed with rehydration. Our population consisted of 15 tall, slim, healthy female volunteers (mean age, wt and ht = 27 yrs, 57kg, 165cm) with initial normal cardiac exam, echo, and history. The study was randomized, placebo controlled, and cross-over in design. Examiners and interpreters were blinded to control vs treatment and furosemide (F) vs placebo (P). Subjects were fluid restricted for a total of at least 5 hrs after breakfast. Each was examined by auscultation and echo prior to and 3 hours after treatment with F or P. After treatment, those with MVP suggested by auscultation or echo were rehydrated and PE and echo were repeated 30 min later. All subjects lost wt; but mean wt loss was significantly (p=0.005) more after F (1.2kg) than after P (0.5kg). An MVP-like murmur was heard in 4 subjects after F and none after P; 2/4 also had echo MVP. Echo suggested MVP in 0/15 before treatment and 7/15 after P and 7/15 after F. LA size decreased more after F(0.3cm) than after P (0.01cm) ($p<0.04$). Mean systolic BP decreased significantly after F but LV dimensions, CO, HR, and diastolic BP remained unchanged. MVP-like murmurs disappeared in all 4 rehydrated subjects(mean wt increase =0.5kg). Rehydration (mean wt increase of 0.7 kg) was also performed in 5/14 who developed MVP-like changes on echo; echo changes resolved in 2/5. Conclusion: MVP-like changes can be induced by mild dehydration in females with phenotypic body habitus of MVP; these changes may resolve with rehydration. Our results may suggest an explanation for variable physical exam findings in individuals with MVP.

THE SIGNIFICANCE OF SUBVALVAR INVOLVEMENT AS A CAUSE FOR MITRAL VALVE INSUFFICIENCY, AS ASSESSED DURING IN VITRO BALLOON VALVULOPLASTY OF RHEUMATIC MITRAL VALVE STENOSIS.

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To assess the mechanism of increase in mitral orificial area during balloon valvuloplasty, 43 rheumatic mitral valves, surgically excised intact were selected and divided on the basis of the leading pathologic feature. Four distinct groups evolved: 1) fibrosis of mitral valve leaflets and commissures (10 valves); 2) fibrosis with calcification of one commissure (8 anterolateral, 7 posteromedial); 3) fibrosis with calcification of both commissures (7 valves); 4) predominant involvement of the subvalvar apparatus (11 valves).

Pictures, X-rays and measurements of the ostia with a conic measure were performed before and after balloon valvuloplasty (balloons up to 38 mm (bifoil 2x19 mm) and pressure up to 4 atmospheres). Valve areas increased with a mean of 165% due to opening of commissures. Twice in group 1 and six times in group 4 a tear occurred in a mitral valve leaflet. In the fibrotic group the tear started near the annulus and proceeded downwards. The lack of annular support may be held responsible for leaflet disruption. The tear in the subvalvar group occurred between the fused chordal columns and proceeded upwards. This damage seems to be a direct effect of the fibrotic involvement of the subvalvar apparatus.

In conclusion: involvement of the subvalvar apparatus in a rheumatic mitral valve has an enhanced risk for laceration of the valve leaflets and, hence, for the development of insufficiency.

MORPHOLOGICAL ANALYSIS OF BALLOON MITRAL VALVULOPLASTY: INTRA-OPERATIVE RESULTS.

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To determine the mechanisms of valvular improvement in percutaneous balloon mitral valvuloplasty (BMV), 9 patients (5 M, 4 F, ages 27-67yrs) with severe rheumatic mitral stenosis (MS) underwent intraoperative BMV prior to surgical commissurotomy (SC) (5pts) or mitral valve (MV) replacement (4 pts). Prevalvuloplasty Doppler and cath MV gradients were 15.1±1.7 and 17.0±2.1 mm, and MV areas were 1.0±.1 and 1.1±.1 cm^2, respectively. Echo score (ES), based on valvular and subvalvular morphology, averaged 9.3±1.2. Intraoperative MV diameter was measured from anterolateral (AL) to posteromedial (PM) commissure pre and post BMV and post SC. Baseline MV diameter was 1.3±.1 cm which increased to 1.6±.1 cm post BMV and 2.5±.2 cm post SC. Mechanisms for valvuloplasty improvement included leaflet stretching (LS) (9 pts), and commissural splitting (CS) (6 pts) primarily of the less diseased leaflet: AL, 3 pts, .30±.2 cm; PM, 2 pts, .15±.05 cm; AL+PM, 1 pt, .8 cm. Low ES was not predictive of success: 4 pts with ES of 9.3±.8 and MVA of 1.2±.2 cm^2 had CS of .13±.02 cm; 2 pts with ES of 14,12 and MVA of 0.5, 0.9 cm^2 had CS of .7 cm, .8cm respectively. These results suggest that successful BMV in rheumatic MS is related to LS and CS. However, the extent of CS was greater with surgical commissurotomy than balloon valvuloplasty, and low ES was not always predictive of favorable valvuloplasty result.

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Hall C, New Orleans Convention Center
Arrhythmias: Diagnosis and Mechanisms

A RANDOMISED TRIAL OF MEDICAL THERAPY FOR VASODEPRESSOR VASOVAGAL SYNCOPE

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There is no proven therapy for vasodepressor vasovagal syncope. This may be diagnosed by prolonged 60° head-up tilt. Scopolamine (S) skin patches and B-blockers have been recommended, but only in open studies. Intravenous Clonidine (C) has blocked the vasodepressor reflex in carotid sinus syndrome. A randomised, double-blind, placebo-controlled, double-dummy, crossover trial of S, C and Atenolol (A) was undertaken. 16 patients were randomised, 8 male aged 62±12 years. There were four six-week treatment periods with active treatment by one of the drugs and placebo (P) for the others with additional triple dummy phase. Daily symptom records were kept. In the fourth week of each period head-up tilt was repeated. Occurrence and timing of syncope were noted.

RESULTS: 13 patients completed the study. 1 patient withdrew with side effects on P, 1 on S and 1 on C.

DRUG	TILTS+VE	TIME(MIN)±SD			SYNCOPE*
S	13	6	33	± 8	1
C	13	6	37	± 9	4
A	13	8	19	± 7	1
P	13	9	26	± 8	3

*during each treatment period

There was no difference between the drugs in the number of positive tilts (Fisher's exact). The mean time of syncope was not different between S and C, but both delayed syncope compared to P ($p<0.03$) and to A ($p<0.001$). Syncope was also earlier with A than with P ($p<0.02$).

CONCLUSION: This trial suggests benefit for C and S in vasodepressor vasovagal syncope. In view of the number of syncopes reported with C, S is recommended as first line therapy. No role is found for therapy with A.